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Review Article

Cardiomyopathy in Pregnancy: A Review Literature

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ABSTRACT

Background : Pregnancy is a physiological process that many women can achieve. Pregnancy in asymptomatic patients with cardiomyopathy is usually well tolerated. However, regardless of the poor prognosis, breastfeeding cannot be accepted in the restricting form of cardiomyopathy. Prior heart events, inadequate results (NYHA with references class III or IV), or advanced left ventricular systolic dysfunction greatly increase the possibility of cardiac symptoms in pregnant women. In spite of intense medical care, clinical conditions can worsen during pregnancy. Although the incidence of cardiovascular disease is present 0.5-4% in developed countries, our knowledge about various cardiomyopathy and pregnancy should be updateable.

Objective : Knowing each type of cardiomyopathy in pregnancy will help the doctor to provide a holistic approach to pregnant women

Discussion : Peripartum cardiomyopathy in pregnancy is the most common type of cardiomyopathy; therefore, a thorough review is needed to give the best outcome for pregnancy. Arrhythmia is most prevalent in hypertrophic cardiomyopathy. Regular monitoring and therapeutic measures should be taken if the arrhythmia is life-threatening for the mother and child. The commonest form of restrictive cardiomyopathy in pregnancy is cardiac amyloidosis.

Conclusion : Our literature provides three types of cardiomyopathies in pregnancy with an example condition for each type that is relevant during pregnancy.

1. Introduction

In general practice, heart disease is expected to be particularly found in pregnant women1. The study identified elevated rates of maternal cardiac adverse effects during birth, including an increased risk of maternal mortality. 15% of all births, mostly for heart failure care, were complicated by hospital admission for cardiac reasons.[1]

Maternal mortality is estimated to be much higher than normal in women with coronary disease and the rate tends to escalate in such a manner that heart disease is the main cause of maternal death in western nations.[49] However, any impact of breastfeeding upon on development of heart-related disease affects pregnancy is relatively unknown.[2]

Cardiomyopathy is characterized as a heart muscle condition that causes an irregular myocardium structurally and functionally without a specific cause.[50] Previous findings have shown that more than 700 births are well prognosed in 500 women diagnosed with hypertrophic subtype, but in high-risk cases, three deaths have been reported. Risk factors include various cases of supraventricular and ventricular arrhythmias, congestive heart failure, and ischemic stroke3. Far less common and frequently related to systemic disease is restrictive cardiomyopathy.[51] In peripartum cardiomyopathy, diastolic activity is uncommon in most patients with a restricted filling pattern consistent with non-segmental hypokinetic in echocardiographic review.[4]

The exact prevalence of restrictive cardiomyopathy pattern in peripartum cardiomyopathy is unknown because of rare cases reported.[4] Although the estimated incidence of cardiovascular disease in pregnancy is present 0.5-4% in developed countries, our knowledge about various of cardiomyopathy and pregnancy should be updated.[5]

Our current literature, therefore, aimed to provide three types of cardiomyopathies. We provide three types of cardiomyopathies in pregnancy with an example condition for each type that is relevant during pregnancy. Peripartum cardiomyopathy in pregnancy is the most frequent type of cardiomyopathy.[4]

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Therefore, a thorough review is needed to give the best outcome for pregnancy. Arrhythmia is the commonest form in hypertrophic cardiomyopathy which need to be regularly monitored and measure should be taken if the arrhythmia is life-threatening for mother and the child. The normal form of restrictive cardiomyopathy in pregnancy is cardiac amyloidosis.

2. Discussion

2.1 Cardiomyopathy and Pregnancy

2.1.1 Definition and Classification of Cardiomyopathy

Cardiomyopathy (CM) is a myocardial condition in which heart muscle structure becomes weakened sufficiently to cause the myocardial abnormality observed in the absence of coronary lession, an increase of blood pressure, heart valvular disease, and also congenital heart disease.[6] In 1996, CM was classified into hypertrophic, dilated, and restrictive cardiomyopathy subgroups by the World Health Organisation (WHO).[7] The cardiomyopathies that happen in pregnant women sometimes aren't well described since the incidences itself are rare.[6]

2.1.2 Epidemiology of Cardiomyopathy

In developing countries, cardiovascular disease is found in 0.5 until 4% of pregnant women.[5] The Kaiser Permanente Health System, the largest non-profit health plan in the United States, with 9.9 million participants, presented data from 2003 to 2014 that classified pregnant women with heart failure.[6] Among these women, 333 (68.2 percent) had PPCM, 34 (6.9 percent) had non-ischemic cardiomyopathy, and 17 (3.5 percent) had HCM.[6] From 2007 to 2011, 1321 women with cardiac disease were included in the Registry of Pregnancy and Cardiac Disease (ROPAC) survey, a voluntary register for pregnancy and cardiac disease run by the Heart Survey Service of the European Society of Cardiology. There were 89 patients with cardiomyopathy which 32 patients related to DCM, 25 patients related to PPCM, 27 patients related to HCM, and 5 with other cardiomyopathies.[2]

2.1.3 Hemodynamic Changes during Normal Pregnancy

During pregnancy and childbirth, marked differences in cardiac output will happen. Each contraction of the uterine can add up to 500 milliliters of mother blood volume due to autotransfusion to uterine blood.[52] The cardiac output will rise by 30 percent during the first stage of labor and up to 50 percent in the second stage due to mother contraction.[9] Cardiac output will enhance the sympathetic surge triggered with anxiety and also pain. Rapid plasma volume changes can contribute to pulmonary edema in women with cardiomyopathy. Epidural anesthesia is also linked to contributing peripheral vasodilatation and intermittent hypotension8. The improvement in stroke volume (SV) can be counteracted by blood loss during childbirth, with a mean blood loss of 500 ml for an uncomplicated vaginal delivery and 1000 ml for a cesarean section8. Cardiac production will increase by 80% over pre-labor values immediately after birth due to auto uterine blood transfusion also IVC congestion relief.[7,8] Systemic venous resistance continues to improve and over subsequent days to weeks the cardiac output normalizes.[5]

Many hemodynamic transitions happened during breastfeeding, which may greatly affect even asymptomatic individuals with cardiomyopathy. Due to physiological anemia, the expanding plasma volume-related breastfeeding is greater than the increase in haemoglobin.[53] Systemic vascular resistance decreases towards the end of the second trimester, which then increases towards the end of birth.[6,7] Concentric remodeling and/or slight eccentric hypertrophy of the heart leads to a proportional increase in both wall thickness and size of the chamber.[8] Both the rise in blood volume and circulating levels of sex steroid hormones are caused by these changes.[8] In particular, progesterone appears to increase protein synthesis and thus cause hypertrophy in heart muscle cells.[8]

2.2 Dilated Cardiomyopathy in Pregnancy

2.2.1 Definition

Dilated cardiomyopathy (DCM) is characterized as left ventricular and systolic dysfunction dilatation without other causes of myocardial dysfunction, such as valvular, congenital, coronary, or systemic diseases.[9] Several causes are known as DCM etiology, but half of all cases are idiopathic10. Idiopathic, myocarditis/infections (viral, Lyme and Chagas disease, or HIV), ischemic heart disease, PPCM, hypertension, infiltrative sarcoidosis, inflammatory or autoimmune (systemic lupus erythematosus), substance misuse due to alcohol, cocaine or amphetamine use, chemotherapy or stroke are the etiology of DCM in women with childbearing.[11]

2.2.2 Hemodynamic Changes in Pregnant Women with DCM

Women already known to DCM have a restriction on accommodating pregnancy, particularly in the time of labor and delivery.[54] In both times, cardiac output is often increased with increased preload, which can worsen heart failure or pulmonary edema.[12] These limitations become more visible during the labor phase when there is a certain significant problem related circulatory system that needs to accommodate increasing oxygen demand by increasing cardiac output.[12] After birth, because of the reduction of the inferior vena cava (IVC) compression by the fetus, there is a rapid change in fluid equilibrium that instantly raises the preload. Patients related to dilated cardiomyopathy can have a certain condition such as exacerbation of heart failure, edema pulmonum, and also arrhythmias.[12] The loss of the low-resistance placenta raises the left ventricle afterload after birth, which could put women with DCM at higher risk of severe hemodynamic deterioration.[54]

2.2.3 Planning for Pregnancy, Delivery, and Long-Term Consideration in Women with Dilated Cardiomyopathy

As pregnancy will change hemodynamic, so women with pre-existing DCM should be noticed if she desires to get pregnant.[54] Women with ejection fraction <30% are classified as World Health Organization (WHO) with class IV classifications contraindicated to get pregnant.[55] Pregnancy will alter hemodynamics, but if they try to get pregnant, women with pre-existing DCM should be noticed from Grewal et al. For females with moderate to severe DCM, progestin-only contraceptive is appropriate, due to the high rate of administration collapse, oral intake should be stopped56. While pregnancy is not advised in some cases, many women have their own decisions. Women can take part in monthly follow-up before gestational week 30 and are prescribed as a routine before delivery twice a month after that. The NT-proBNP value should be used and regularly tracked during the first visit.[12] To consider pulmonary strain, non-invasive testing such as echocardiography may also be effective.[12]

Women who have dilated cardiomyopathy are likely to deliver by normal vaginal method on the day of birth and the cesarean delivery should be reserved when obstetric signs and cardiac dysfunction appeared.[12] Consideration should be extended to anesthetic medications to prevent unnecessary myocardial depression caused by anesthetic and to minimize the inherent activation of the sympathetic involved in the labor.[57]

The third trimester and early postpartum cycles should be monitored by doctors since there can be an accelerated hemodynamic transition.[56] Cardiac decompensation, such as deteriorating cardiac

failure, may precipitate certain adjustments. But by that time, the follow-up is not halted and there may be a late result on the diseased ventricle that may develop months after completion of the birth.[58] An impairment of the ejection fraction and contractility can be associated with the late results. It required the discontinuation of optimal medical care for HF due to contraindications and/or the choice of a patient during breastfeeding and/or lactation.[12]

2.2.4 Acute and Chronic Heart Failure Management in Women with Pregnancy and DCM

In the acute setting, the condition should be divided into stable and unstable conditions. Intravenous diuretic and vasodilator drugs such as nitroglycerin safely enough to use in pregnancy especially in the acute decompensation period.[12] Pregnant women with unstable hemodynamic may use dobutamine and milrinone. In that condition, doctors should have a delivery plan, a cesarean delivery is preferred for unstable condition.[59] After delivery in the post-partum period, lactation should be avoided for the unstable patient. It is different from an unstable condition, a stable patient may be addressed to lactation with preferences and medication safety profile and initiation of heart failure management such as ACE inhibitors, ARBs, beta-blockers, diuretic, or digoxin.[60]

The aims of medicinal treatment during breastfeeding are close to those of non-pregnant women in terms of chronic illnesses. A significant point remains the continuation of treatment. Treatment with ACE inhibitors, ARBs, and inhibitors of angiotensin receptor-neprilysin (ARNI) is contraindicated in pregnancy due to teratogenic and harmful effects on the growth of the fetal kidney, and also spironolactone (FDA category C).[60] In patients with heart disease during breastfeeding, it is safe to treat hypertension with amlodipine.[61] Particularly if asymptomatic, pregnant women can proceed with β- blockers (BB) for the prevention of persistent heart disease. If necessary, the use of hydralazine and nitrates as a vasodilator is necessary as a cure for heart failure.[12]

2.3 Peripartum Cardiomyopathy

Examples of dilated cardiomyopathy include peripartum cardiomyopathy (PPCM) (DCM). This is a rare disease characterized by systolic dysfunction in early or late pregnancy and even postpartum periods.[13] In the 19th century, PPCM was known as heart disease, but the term "peripartum cardiomyopathy" was not used until 1971, when Demakis et al. published a broad case series and established the first diagnostic criterion which is the progression of heart failure within five months of delivery.[14]

2.3.1 Definition of PPCM

PPCM is a condition of exclusion attributable to related left ventricular systolic dysfunction in women who present with HF which should be treated where no other cause is apparent. Therefore, the European Society of Cardiology Working Group's 2010 Heart Failure Association already revised the term of PPCM to “an idiopathic cardiomyopathy presenting with HF secondary to LV systolic dysfunction towards the end of pregnancy or in the months following delivery, where no other cause of heart failure is found”. The criteria for diagnostic shows that LVEF is <45 percent and ventricular dilatation can occur or sometimes within normal limit.[15]

2.3.2 *Epidemiology of PPCM*

The highest prevalence of PPCM is in various ethnic groups in African Americans (1 in 1421).[16] The incidence can be different in geographic regions of the world14, in Nigeria the incidence is estimated as 1 in 100 deliveries[17], 1 in 300 deliveries in Haiti[18], and Japan about 1 in 20,000[19].

2.3.3 Risk Factors

There is a meta-analysis study of 22 reports, which study included 979 patients with PPCM, stated that 22 percent of women with PPCM had pre-eclampsia, relative to an overall global baseline prevalence of 5 percent, and 37 percent had other hypertensive disorders.[20] Seven percent to 14.5 percent of PPCM patients from the United States record multi-gestational pregnancies.[20] In 9 percent of women, a meta-analysis of 16 PPCM studies found an overall prevalence of twin pregnancies relative to the national average of 3 percent. Half of the PPCM cases occur in women > 30 years of age, and 1 study recorded that > 40 years of with odds ratio 10 to contract the disease relative to women < 20 years of age[20].

2.3.4 Pathophysiology of PPCM

The etiology of PPCM isn’t fully understood and is sometimes multifactorial.[15] The present theory supports a "two-hit" hypothesis of PPCM pathogenesis, whereby cardiomyopathy in women with an inherent predisposition is triggered by vascular insult exacerbated by anti-vascular or hormonal effects of some phases which are late pregnancy and also the early postpartum phase.[13]

Any PPCM cluster events in families have long been observed.[21] Subsequently, in 172 women with PPC, gene sequencing associated with DCM observed 26 variants, 65% related TTN; a titin-encoding gene.[22] Variation of TTN substantially correlates with mutations suspected to cause DCM. In comparison, the occurrence of a TTN mutation against others expected lower LVEF at 12 months (LVEF at 12 months 44 percent v 54 percent overall, P=0.005; LVEF at 12 months 38 percent v 52 percent among black race patients, P=0.04).[22]

A 2007 research indicated that prolactin plays a part in the pathogenesis of PPCM, a protein hormone secreted by the anterior pituitary. TAT3 is triggered during breastfeeding and the postpartum phase in the normal maternal heart[13]. Via unexplained mechanisms to express cathepsin D, an enzyme that cleaves prolactin into a 16 kDa segment that causes endothelial apoptosis and capillary drop out, oxidative stress stimulates cardiomyocytes. There was an elevated expression of 16 kDa prolactin in their cardiomyocytes during mouse pregnancy, which lacked STAT3[13]. This mouse had greater coronary capillary dropout, increased concentrations of ROS, and the PPCM phenotype was also seen. Subsequent research by the same group has shown that 16 kDa prolactin exerts cardiotoxic effects by upregulation of microRNA-146aa (miRf146a).[23] The 16 kDa fragment triggers the release of exosomes containing miRf146a and other microRNAs from endothelial cells. MiRf146a blocks many pathways that contribute to cardiomyocyte death, including Erbb4, Nras, and Notch1. The high circulating miRf146a normalizes after bromocriptine administration.[13]

Possible shared pathophysiology is indicated by the high prevalence of preeclampsia in women with PPCM. Soluble fms-like tyrosine kinase receptor 1 (sFlt-1) is a placenta-secreted anti-angiogenic enzyme in rapidly rising levels at the end of pregnancy. Vascular endothelial growth factor (VEGF) and placental growth factor (PlGF) are sequestered by sFlt-1 and are considered to be the primary driver's key pathophysiology in pre-eclampsia.[24] In addition, sFlt-1 levels in women with preeclampsia correlate with global longitudinal pressure and increased mass LV25. In normal pregnant women, SFlt-1 levels decrease quickly after childbirth (after eliminating the placental source of sFlt-1) but appear greater than the normal amount in PPCM patients.[26]

2.3.5 Clinical Presentation and Diagnosis PPCM

The majority of women with PPCM, usually in the first month after childbirth, are diagnosed after delivery. Most women have signs

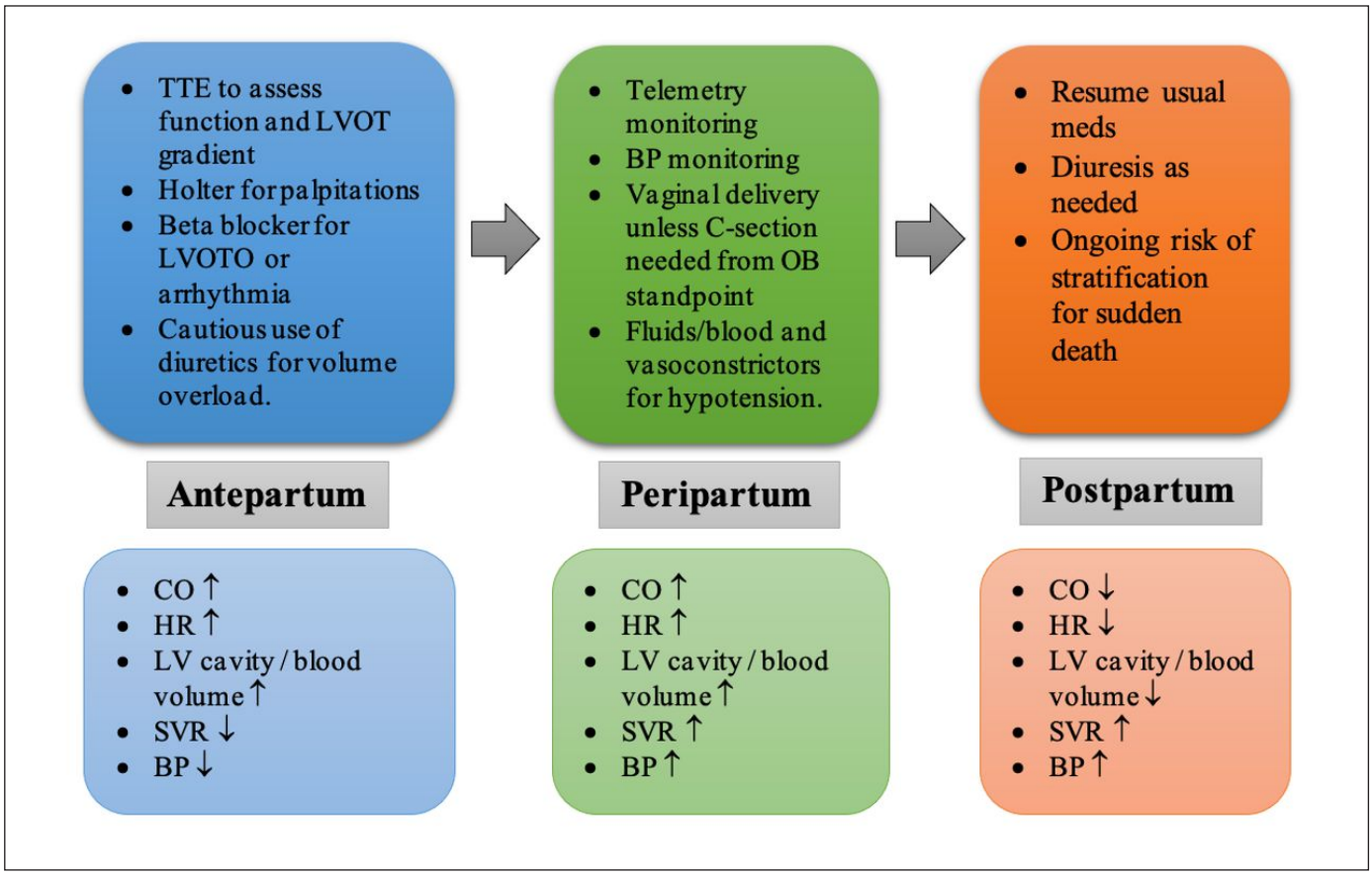

Figure 1. Management of Hypertrophic Cardiomyopathy in Pregnancy. Follow-up patient comprehensively during pregnancy. The target of hemodynamic is seen in the picture and needs to be evaluate thoroughly during antenatal care and postpartum.[48]

and symptoms of HF, including shortness of breath, fatigue, orthopnea, chest tightness, edema, and paroxysmal nocturnal dyspnoea. Physical testing has also indicated tachypnoea, tachycardia, elevated JVP, pulmonary rales, and peripheral oedema[15] A left or right side S3 gallop (or both) can be audible, but in normal pregnancy, an S3 gallop may also be present.[13] Sinus tachycardia and nonspecific ST-T wave disturbances can be revealed by electrocardiography, as well as left ventricular hypertrophy and left atrial enlargement. Vascular congestion, pulmonary edema, and cardiomegaly can be seen in chest X-rays.[27] In any suspected case of PPCM, echocardiography should be conducted as the LVEF is normally < 45 percent or fractional shortening < 30 percent and the LV end-diastolic dimension is > 2.7 cm/m2 .[28] Intracardiac thrombus can occur, and especially when LVEF is severely reduced, the LV apex should be clearly visualized.[29] If the echocardiogram is ineffective, cardiac magnetic resonance imaging offers precise ejection fraction and chamber measurements, but gadolinium is excluded during breastfeeding.

2.3.6 Outcome and Prognosis Complication PPCM

In order to ensure fetal protection, the management of PPCM involves special treatment. Many HF drugs are consistent with breastfeeding after birth.[15] The increased prevalence of LV thrombosis and systemic thromboembolism has been documented in women with PPCM and the hypercoagulable state of pregnancy and the early postpartum period.[30] Warfarin crosses the placenta for indications other than artificial heart valve anticoagulation and is stopped during breastfeeding. No low-molecular-weight heparin crosses the placenta and can be used during breastfeeding. Warfarin and low-molecular heparin are both considered healthy for lactation.[62]

2.3.7 Management of PPCM

The increased prevalence of LV thrombosis and systemic thromboembolism has been reported in women with PPCM and the high coagulation state of pregnancy and the early postpartum period.[30] When the LVEF is <30%, anticoagulation is recommended by the American Heart Association[28], while the European Society of Cardiology recommends using LVEF <35% as the criterion.[31] Warfarin crosses the placenta for indications other than artificial heart valve anticoagulation and is stopped during breastfeeding. The placenta is not crossed by low-molecular heparin and should be used during breastfeeding.[32] With lactation, both warfarin and low-molecular-weight heparin are considered stable. A retrospective registry of 115 German PPCM patients indicated that bromocriptine was associated with a higher rate of progress in LVEF in comparison to normal treatment, but there was no substantial change in overall recovery rates.[33] It is important to explore with the patient the consequences of not breastfeeding due to bromocriptine. A randomized, double-blind, placebo-control trial (REBIRTH [Randomized Assessment of Bromocriptine in Myocardial Recovery Therapy]) evaluate the impact of bromocriptine on myocardial recovery in 200 women with myocardial recovery and clinical outcomes related to PPCM in the United States and Canada has been proposed by the IPAC group and is under evaluation. A Class IIb recommendation for the use of bromocriptine is included in ESC recommendations (Figure 1).

Intravenous vasodilators such as nitroglycerin can be needed in the case of acute decompensated HF during pregnancy. The 7 women treated with dobutamine had worse results, but selection bias may have occurred in this small study. PPCM triggers a total of 60 percent of

cardiogenic shock cases during or immediately after birth.[34] Temporary mechanical circulatory aid with intra-aortic balloon drive, percutaneous ventricular assist device therapy, and extracorporeal membrane oxygenation has been successfully utilized in PPCM and should be considered early in patients with hemodynamic instability amid inotropic help.

When and how the delivery of women presenting with PPCM during pregnancy should be discussed with the patient and organized by a cardio-obstetrics team of specialists from obstetrics, and many other related fields.[35] Hemodynamic disruption could cause early delivery despite medical therapy (or termination if before fetal viability). If there are obstetric grounds for cesarean surgery, healthy patients are supplied vaginally.[15]

2.4 Hypertrophy Cardiomyopathy in Pregnancy

2.4.1 Definition and Epidemiology

The diagnosis of hypertrophic cardiomyopathy (HCM) is based on a hypertrophied, non-dilated left ventricle without an exact anatomical or physiological finding and, in the absence of another cardiac or non-cardiac finding, is detected by echocardiography or magnetic resonance imaging (MRI).[63] In this case, echocardiographic epidemiological tests found an incidence of 1 case per 500 people in the general population, but when clinical and genetic diagnoses are mixed, cases are identified in 1 case per 200 people. An approximate 750,000 individuals can be impacted by HCM in the United States. However, only a handful of them has been diagnosed with the condition (about 100,000), sometimes by means of non-invasive imaging when encountered or when there is a low ejection fraction which makes the symptom worsen.[36]

2.4.2 Clinical Imaging

The characterization of the HCM phenotype is split into two general distinctions: the pattern of the hypertrophic wall and the outflow obstruction pattern. The discrepancy between these two technologies was focused on nearly 50 years of echocardiographic imaging. In certain patients, high-resolution tomographic MRI may offer a more accurate evaluation of left ventricular hypertrophy and even improve stratification assessment by detecting myocardial fibrosis in vivo. The thickness of the left ventricular wall is 15 mm or more (mean, 21 mm) in most clinically diagnosed cases, although large thicknesses (30 to 50 mm) are often known to be hypertrophic wall trends. A differential diagnosis of chronic hypertension or physiological athlete's heart also includes borderline thickness (13 to 14 mm). In certain gene carriers, however, some left ventricular wall thickness is compatible with the clinical continuum of HCM, including standard measurements. HCM is primarily an obstructive condition, with 70% of patients with mechanical impedance to the production of left ventricular outflow (gradients ≥30 mm Hg) at rest or with physiological agitation such as mild strength movement activity. Outflow obstruction is normally caused by mitral-valve motion in systolic and septal interaction due to flow drag observed in mitral regurgitation.[36] (figure 1)

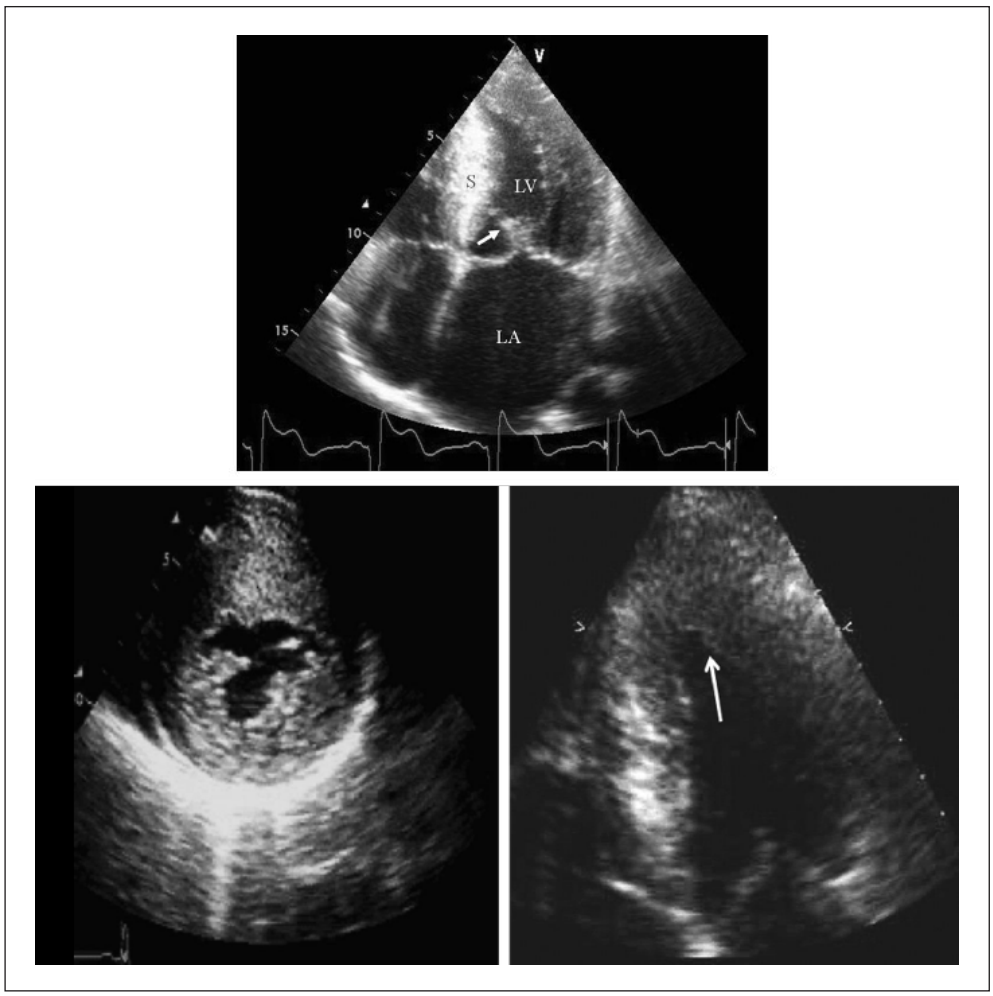

Figure 2. Hypertrophic cardiomyopathy is primarily diagnosed by echocardiography

2.5 Arrhythmias in Pregnancy

2.5.1 Definition and Pathophysiology

Hypertrophic cardiomyopathy (HCM) is described by increased LV wall thickness that can't be clarified by causes of excessive loads, such as hypertension or valvular disease. Many women who have good functional status and are asymptomatic of HCM can tolerate pregnancy and the resulting rise in plasma volume. Adverse effects, though, will occur. Counseling for preconception and careful observation during breastfeeding is important. While no maternal deaths were recorded in this registry, heart failure and/or ventricular tachyarrhythmias occurred in many women with HCM. Before birth, women should be closely monitored because signs of heart disease or reduced functioning status are related to elevated risk during pregnancy.[37]

Plasma volume and also cardiac output rise during breastfeeding. Increased cardiac output is achieved by a larger number of strokes in the first and second trimesters, while the heart rate drops later in pregnancy. The added pregnancy volume load induces ventricular cavity enlargement, which may potentially minimize the obstruction of the LVOT. The raising cardiac output, however, appears to offset this impact, and with progressing gestation, the LVOT gradient also increasing. The same loading volume raises the left atrium distension and hence the possibility of atrial fibrillation. Volume fluctuations and elevated heart rhythm are not well handled in the sense of diastolic disease, aggravating the effects of dyspnea and lowering the threshold for developing left heart failure. At birth, a further rise in cardiac production is secondary due to blood auto-transfusion from the contracting uterus and elevated catecholamine levels. Changes in heart rate are often secondary to blood loss, pain, and stress, while expulsion helps decrease venous return during contraction. All of these physiological changes contribute to a rise in the LVOT gradient and shorten the duration of diastolic loading, thereby increasing the risk of pulmonary oedema.[38]

2.5.2 Clinical Presentation

Diastolic left ventricular dysfunction is invariably present, though some patients often experience systolic dysfunction later in the course of the condition. Mitral regurgitation is frequently synonymous with LVOT obstruction, often due to mitral valve systolic anterior motion (SAM), whereas inherent deficiencies in the mitral valve system often contribute to regurgitation of the mitral valve. Dyspnoea and chest pain, combined with the pathophysiologic effect of diastolic dysfunction, LVOT obstruction, MR, and myocardial ischemia, are the most common symptoms. Atrial fibrillation is the most common arrhythmia, and the high likelihood of thromboembolism is poorly tolerated. There is a growing risk of sudden death in people with a family history of sudden death, ventricular tachycardia, syncope, a blood pressure response due to exercise, and severe hypertrophy. An implantable cardioverter-defibrillator (ICD) will minimize the risk of accidental death in this type of patient.[39]

It is doubtful that a new arrhythmia substrate will be produced by the pregnant state; any physiological changes would render a pre-existing substrate capable of sustaining an arrhythmia. A bigger heart can tolerate re-entry more efficiently and it is well understood that arrhythmogenic mechanical elongation is. Pregnancy may also be the explanation for the onset of arrhythmia. Many tachycardia episodes are caused by ectopic beats and the incidence of arrhythmia episodes may rise during pregnancy, in line with the increased propensity for ectopic surgery.[40]

2.5.3 Management of Ventricular Arrhythmia due to Hypertrophic Cardiomyopathy in Pregnancy

During pregnancy, women taking beta-blockers should continue to take the drug. For any woman who experiences problems during breastfeeding, it is often recommended that she should start a beta-blocker and, if possible, prescribe diuretics. A beta-blocker should be considered if there is more than mild LVOT obstruction or more than 15 mm of septal thickness, in order to minimize the possibility of pulmonary congestion.[41]

Beta-blockers are also useful for regulating and lowering the occurrence of ventricular arrhythmias in atrial fibrillation. As there is adequate experience of its use in breastfeeding, metoprolol is the recommended beta-blocker; however, bisoprolol can also be used. The newborn should be treated for bradycardia and hypoglycemia after delivery.[38]

Verapamil may be used during breastfeeding, but due to the possibility of fetal atrioventricular blockage, caution is required. Since it can cause fetal thyroid disorders, amiodarone should not be used unless strictly necessary. During breastfeeding, Sotalol may be used. Finally, in the event of a maternal emergency, any drug that is toxic to the child should not be refused. Atrial fibrillation is poorly tolerated and, either chemically or electrically, involves prompt cardioversion to preserve sinus rhythm. Electrical cardioversion during pregnancy is deemed healthy, but regular monitoring of the fetal heart rate and the prospect of an immediate cesarean operation should be given when the fetus is at a viable age.[42]

2.6 Restrictive Cardiomyopathy in Pregnancy

2.6.1 Definition and Epidemiology

In restricted cardiomyopathy, nondilated left or right ventricles with diastolic dysfunction or both systolic and diastolic dysfunction are characterized by nondilated left or right ventricles without any other associated cardiac or systemic diseases. A heterogeneous community of myocardial diseases that differ in clinical appearance and another clinical subset of restrictive cardiomyopathies are. Restrictive cardiomyopathy is complicated by enhanced myocardial stiffness and decreased elasticity as recoil results in recent decreased ventricular loading and ejection fraction.[43]

Genes that are active in hereditary RCM specifically encode cardiac sarcomere proteins. There is no maternity data available for RCM patients. This is because RCM is very unusual and the RCM treatment path is always serious with a bad prognosis.[44]

2.6.2 Clinical Imaging

In restricted cardiomyopathy (RCM), a patient with normal or near-normal systolic activity and signs of diastolic dysfunction with a restrictive echocardiography filling pattern should be suspected of being diagnosed. 2-dimensional and Doppler echocardiography are important for the assessment of diastolic dysfunction and the differentiation between patients with RCM and patients with other restricted physiology due to constrictive pericarditis. The echo indicates concentrated thickening of the free wall and septum of the LV and suggests anomalies of geographic wall displacement in non-coronary delivery and aneurysms. In restricted cardiomyopathy, the strain phenomenon corresponds to the deformation of the myocardium at contraction with any percentage length change. Strain imaging has been tested by tissue Doppler imaging in the early stages, but non-Doppler speckle-tracking strain imaging has become a common method with the aid of non-angle-dependent measurements.[45]

2.7 Cardiac Amyloidosis in Pregnancy

2.7.1 Definition and Pathophysiology

This is the most prevalent form of restrictive cardiomyopathy by far, and when explaining restrictive cardiomyopathy, the overwhelming majority of the study relates to cardiac amyloidosis. This form of cardiac activity, however, induces both diastolic and systolic left ventricular dysfunction, which is most commonly found in moderately dilated cardiomyopathies. Interstitial penetration of the atria and ventricles while the heart is involved leads in the more advanced situations to a solid and 'rubbery' quality of the myocardium. Systemic amyloidosis is a protein metabolism condition in which abnormal extracellular protein content is accumulated in tissues and organs. This produces substantial morbidity and is typically fatal. Light chain amyloid also involves the heart, leading to congestive heart failure.[46]

This amyloid infiltration mechanism is responsible for the marked thickening of the echocardiographic vision of the left and right ventricular walls, regular or reduced LV cavity size, and decreased diastolic and systolic LV function. The coronary intramural arteries and the conductive system are often infiltrated and this accounts for the electrocardiographic irregularities found in the first-degree A-V block. The endocardium may also be compromised and can be associated with the overlying thrombus. Amyloid heart infiltration often leads to focal or diffuse valve thickening, but clinical valve dysfunction is uncommon.[44]

2.7.2 Clinical Presentation

The biopsy will only specifically classify cardiac amyloidosis and the echocardiographic features of both types can be distinguished. Amyloid penetration echocardiographic findings consist of: I thickened RV, (ii) thickened LV walls, (iii) 'granular' or 'sparkling' (ground glass) myocardial appearance, (iv) normal or minimal LV cavity size, (v) expanded atria, and (vi) decrease LV systolic and diastolic function.[46]

2.7.3 Management of Cardiac Amyloidosis in Pregnancy

Volume management (diuretics/salt restriction) and arrhythmia management are primarily involved in the cardiac-specific treatment of all types of amyloidosis. Usually used in HF, neurohormonal antagonists are often poorly tolerated and detrimental. Beta-blockers, ACE inhibitors, and ARBs, in particular, frequently contribute to hypotension (due to autonomic instability and the existence of a narrow left ventricular cavity with an inability to raise the volume of stroke in response to vasodilation) and beta-blockers often intensify bradyarrhythmias. Digoxin binds to amyloid fibrils, contributing even at usual circulating levels to the risk for digoxin toxicity, which should be usually avoided. Pregnancy in this patient is not appropriate, so terminating is the choice of care and multidisciplinary consultation is compulsory.[47]

3. Conclusion

Pregnant women with cardiomyopathy may have a form of cardiomyopathy that is dilated, hypertrophic or restricting. Peripartum cardiomyopathy, which is a dilated type of cardiomyopathy, is the most prevalent type of cardiomyopathy in pregnancy. In general, except for restrictive cardiomyopathy, cardiomyopathy that happened during pregnancy may be safely handled medically with no risk to the mother or fetus. Where practicable, medications should be prevented in the first trimester and the treatment preference should indicate the safety record in pregnancy as well as the precise type of cardiomyopathy being treated and any underlying heart condition associated with it. Drugs should be supplied at the lowest effective dosage and closely supervised for the mother and her baby during pregnancy.

4. Declarations

4.1. Ethics Approval and Consent to participate
Not applicable.

4.2. Consent for publication
Not applicable.

4.3. Availability of data and materials
Data used in our study were presented in the main text.

4.4. Competing interests
Not applicable.

4.5. Funding source
Not applicable.

4.6. Authors contributions
Idea/concept: AADA. Design: AADA. Control/supervision: AR. Data collection/processing: AMZI. Extraction/Analysis/interpretation: AMZI. Literature review: AMZI. Writing the article: AMZI. Critical review: AR, HM, NK. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

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